A CASE OF APRAXIA, WITH AUTOPSY[1]

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Until comparatively recently, by apraxia was meant the inability to use an object because its nature, or its use, was not recognized (Küssmahl and Starr (48)). Since Liepmann's exhaustive studies on the subject, however, apraxia takes on a different signification. It is a condition entirely independent of agnosia, or the failure to recognize an object.

According to Liepmann's (23, 24) quite recent opinion, the apraxic recognizes the object and its use, is able to move the limbs readily, indicating that the innervation of each limb is intact; but purposeful movements by the affected limbs are impossible. This, at least, is the definition for pure motor apraxia.

The description of apraxia by Pick (40), Van der Vloet (44), Margulies (28), and von Monakow (34), and the views held by Marie (29), especially, are not entirely in accord with this definition. Psychical disturbances in apraxics; the appearance of apraxia in certain forms of insanity; the element of inattention and lack of concentration so often observed in the apraxic subject, all suggest the possibility of the presence of other elements in the phenomena of apraxia, than are included in Liepmann's definition.

The whole subject is a difficult and complicated one, and the following case, while exhibiting certain symptoms in common with those of some of the reported cases, is generally unlike any case in the literature at my disposal.

The patient, J. C. G., aged 55 years, married; by occupation a railroad builder, and one-time member of the Canadian

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Parliament, was admitted to the Philadelphia Home for Incurables, July, 1906, complaining of loss of vision.

The family history was entirely negative. There was no previous history of illness, excepting the ordinary diseases of childhood. He had always worked hard; had been exposed to the elements as a young man, frequently sleeping out at night; and had always been a moderate, and at times excessive, user of alcohol. There was no history of specific disease.

His present disease began with failing vision, about three years before his admission to the Home. At the end of two years it had progressed so that he was unable to read.

Dr. David Webster and Dr. J. F. Terriberry (52) reported this case before the New York Ophthalmological Society in 1906.

Dr. Webster stated that the patient came under his observation August 28, 1905, at which time there was little more than perception of light. When the hand was held before the eyes he knew there was something there, but did not recognize it. He was unable to count fingers at any distance, or in any position, but he did not run into objects when walking about the ward. Dr. Webster believed that he actually saw objects, but could not distinguish their form; in other words, that he was suffering from "visual astereognosis." At that time the ophthalmological examination revealed no cause for the blindness.

Dr. Terriberry reported upon the neurological conditions at the same meeting. At this time the patient was unable to locate touch, which was, however, perfectly perceived. His sense of motion was also disturbed, and he was unable to execute any movement correctly. The muscular sense was absent in all the extremities. There was no disturbance of the sense of heat and cold; and taste, smell and hearing were normal. There was no evidence of palsy.

The vision continued to fail until, at the time of his admission to the Home, he was unable to distinguish objects, and the blindness appeared to be absolute, as he denied seeing the hand, or a flame, when it was held before his eyes.

He had had no difficulty in using his arms apparently, as he always dressed and fed himself, until some time in 1904 (over a year before admission) when his wife noticed that, if she took his left hand to lead him, he did not recognize that she held it. He gradually gave up the use of the left hand on the balusters in going up and down stairs, and in eating and dressing.

PRESENT CONDITION: *Vision.*—Dr. William Campbell Posey examined his eyes shortly after his admission, with the following results:

"Eyes appear fixed and expressionless, the patient rotating them but rarely, in marked contrast to the searching, rolling movements so frequently observed in the blind. Winking is performed rather less frequently than is normal (Stelwag sign). When requested to regard an object, the patient stares straight ahead, even though the object toward which he is told to direct his gaze is at the side, and the width of the palpebral fissures is somewhat increased on both sides (Dalrymple sign).

"There are no palsies of the extraocular muscles, although it is difficult to make the patient turn his eyes in the direction required. Both corneas are clear, and the eyes are free from any traces ot recent or old inflammation. Both pupils are 5 mm. in size, and are quite active to direct and consensual light stimuli. This reaction is obtainable in diffuse sunlight, and to a much less degree from the light of an ophthalmoscopic mirror. The Wernicke pupillary inaction sign was searched for, but was not present, although the pupils reacted more readily to stimulation of certain parts of the retina than others; but these reactions were not from symmetrical portions of the retina, and were probably dependent upon a varying degree of atrophy of the optic nerves, and also upon the impossibility of always confining the light stimuli to definite and circumscribed retinal regions.

"The media are clear, and the optic nerves exhibit the early changes of a simple atrophy. As yet, however, there is no true atrophy, the nerves being dull red-gray, and possessing the appearance so frequently seen in men in middle life who have abused tobacco and alcohol, or who have cerebrospinal sclerosis. The blood currents are also well maintained, and the retina seems well nourished, and there is not sufficient ophthalmoscopic cause for the total blindness which is apparently present.

"Loss of vision appears absolute, as the patient denies seeing the hand when it is held close before his eyes, or even a flame, and that the blindness is not feigned is evidenced by the fact that it is not possible to obtain compensatory movements of the eyeballs by the use of prisms. On account of the absence of total atrophy, the apparent preservation of the functioning power of the retina, and the undoubted active reaction of the pupils to light stimulus, it would appear that the cause of the blindness is central, and not peripheral, and that the lesion producing it must be posterior to the center which regulates pupillary movements."

SENSORY CHANGES: *Sense of Position.*—The patient was unable to tell whether he was standing or sitting, although occasionally, when this symptom was tested, he answered correctly. He failed to recognize the position of any of his limbs, or that they were being moved.

Location.—He was incapable of locating, anywhere about his body, the prick of a pinpoint, light or deep pressure, or heat or cold.

Sensation.—Tactile sensation appeared to be lost in the left hand, but was present in the right hand, and elsewhere about the body.

Pain Sense.—While examinations of the pain sense were rather unsatisfactory, it was probably preserved. Pricks with the sharp points of the esthesiometer generally were followed by an expression of irritation. The right hand was withdrawn, but the left hand made no movement whatever.

Temperature Sense.—He confused heat and cold on the left hand, but elsewhere about the body the temperature sense seemed to be unimpaired.

Pressure Sense.—He was unable to distinguish between light and deep pressure anywhere.

Stereognostic Sense.—He was totally unable to recognize objects by the sense of touch. He recognized no object placed in either hand.

MOTOR SYMPTOMS: The general muscular power seemed to be fairly good. The dynamometer registered 40 on the right, and 30 on the left. (He was right-handed.) There was no palsy of any of the limbs, and he was able to walk, although his gait was peculiar and will be described later.

APRAXIA: He presented a few motor manifestations which, in all probability, were the result of apraxia, although this conclusion must be somewhat guarded, as the complete loss of the sense of location of the limbs and the body generally, the complete failure to recognize objects by the sense of touch, and the loss of the sense of movement, complicated the study of these symptoms. Another symptom which still further complicated the study of the case, was the behavior of the left arm. It was held in a slightly flexed position, the fingers semi-flexed most of the time, and rarely or never was it moved voluntarily. This was not due to paralysis, as the grasp was fairly good, and at other times the arm was observed to move freely, apparently without any sign of paralysis (Seelenlähmung).

Left Hand.—When asked to place to his ear a watch held in the left hand, no movement whatever of the left arm followed, but instead the right arm was stretched forward, and the right hand grasped either his knee or his coat, while at the same time he believed that he was placing his left hand to his ear.

When asked to squeeze the hand of the examiner with his left hand he was successful at times, but more often distorted, or irregular movements of the right arm followed, or he grasped some portion of his body with the right hand, and squeezed violently.

When a fork was placed in his left hand and he was requested to feed himself, the left arm remained motionless, or moved slightly only.

When told to touch his right hand with the left hand, only a slight movement in the left hand resulted, while the right hand groped aimlessly, as before described. Or, he would rise to his feet, without moving either hand, and believed he was making an effort to obey the command.

On the other hand, on several occasions he was able to take from his trousers pocket a handkerchief and wipe his nose with it voluntarily or when requested to do so, with either hand (reflex movement).

Right Hand.—The right hand was also the seat of disturbances of volitional acts. When a watch was placed in his right hand, and he was told to carry it to his ear, while at times this was successfully accomplished, he more often carried it to his mouth, and went through the motions of chewing; and this, in spite of the fact that he recognized perfectly that it was a watch that he was to place to his ear.

In feeding himself a banana he invariably put the banana to his chin instead of his mouth, if unassisted. In the absence of ataxia this is interpreted as being an apraxic movement.

He was unable to correctly use the hand for the purpose of dressing or eating.

In masticating his food the bolus was not moved around the buccal cavity in the usual manner, the jaw being moved rapidly up and down, and the food was often retained in the mouth a long time before he swallowed it.

When asked to grasp the hand of the examiner with his left hand, he occasionally succeeded, and at such times he would not relax his hold and apparently involuntarily, in fact, grasping more and more tightly, as long as the hand of the examiner remained within his grasp. This was undoubtedly a manifestation of perseveration, of the tonic variety described by Liepmann.

Gait.—The gait was peculiar. Short steps were taken, usually to one or the other side, rather than forward. From this he never varied, even when every precaution was taken to show him that there was no danger of his bumping into anything; in other words, to eliminate the influence of the loss of vision.

Writing.—When a pencil was placed in his right hand, and he was asked to write his name he moved the pencil up and down, from right to left, without forming any letters. He was entirely unable to make any movements when the pencil was placed in his left hand.

Mental Condition.—His intellect seemed to be fairly good. His memory was excellent and he discussed general matters cleverly and intelligently, apparently understanding everything that was said to him. He was, however, impatient, irritable and unreasonable, so that, unfortunately, the examination could not be carried out in the desired detail.

Reflexes.—The tendon reflexes of the arms were present on both sides. There was no Babinski sign or ankle clonus. The plantar reflex was exaggerated on both sides. The patellar reflexes were present, and probably exaggerated, but the rigidity of the limbs prevented much movement of the legs on the thighs.

Station.—With the feet together he stood perfectly.

Contractures.—The left hand was held in a slightly contracted position, as above indicated; that is to say, partial flexion of the forearm on the arm, and semi-flexion of the fingers. This could be passively overcome almost entirely, but there developed at the same time a tremor of both arms, especially of the left. There was also a slight but distinct rigidity of both legs and the right arm, but to a less extent than in the left arm.

Examination of the ears, made by the late Dr. W. G. B. Harland, gave the following results:

"Cerumen in both ears, but causing him no discomfort, and I doubt that removal of the masses would change the findings much. A more important source of error is found in the unreliability of his statement. We can say positively that he hears plainly the ordinary voice, but cannot hear higher tones, this defect being more noticeable on the left side. Deafness for low tones is also worse in the left ear, as shown by the tuning forks.

"He does not know that he is deaf, nor does he suffer from tinnitus. The results of the examination are what we might expect in an old man with a mild degree of middle ear disease, with secondary nerve involvement."

The tongue was protruded straight on command, and in the median line. It was tremulous and tooth-indented.

The left hand was swollen, and the left forearm was generally wasted slightly.

A diagnosis of bilateral cysts in the occipito-parietal region was made, an exploratory operation advised, and on February 1, 1907, Dr. Edward Martin opened the skull in the right occipito-parietal region. The pia was much thickened, and what appeared to be an area of softening was found. The patient made a good recovery from the operation, but the symptoms remained unchanged.

On April 4, 1907, efforts to overcome the contracture of the left arm and hand appeared to give pain. The left arm was very tremulous, and the rigidity generally seemed to have increased. His mentality appeared to have suffered, and it was impossible to make any satisfactory tests of his condition on account of his extreme irritability.

On May 10, 1907, the contractures of the left hand had increased, and the fingers were pressed forcibly into the palm of the hand. There was a Babinski sign on the left side at this date. There was no facial asymmetry. He was confused and violent, and his passages were involuntary.

There was at no time any vomiting, headache, or pain elsewhere in his body.

At this examination his sense of taste was markedly altered. He could not tell salty foods from sweet, and could not tell the consistency of food when it was placed in his mouth.

He gradually failed mentally, became at times excited, refused food, and died of exhaustion.

Autopsy.—At the autopsy there was an intense internal pachymeningitis on the right side, which was not observed at the time of the operation. The inner surface of the dura was covered with a thick plastic exudate, extending over the entire surfare of the brain on the right side. The dura was everywhere adherent to the skull, but was readily dissected away, with slightly more difficulty over the site of the operation. The bony plate which had been removed at the time of the operation and replaced was depressed about 3 millimeters below the level of the skull. Bony union seemed to have occurred in places, but there were two areas at the site of trephining where no closure by bone had taken place.

A necrotic area was observed on the right side of the brain, involving the cortex in the region of the occipito-parietal fissure, extending 3 centimeters in front of, and 15 millimeters behind this fissure, and laterally 45 millimeters from the longitudinal fissure. A cross-section in this area showed that the cortical layer was very narrow—about one millimeter in thickness. In the right occipital lobe the cortex was not half the normal thickness. On the left side it was macroscopically apparently normal.

The brain was first placed in formalin, and then in Müller's fluid. It was divided horizontally into six approximately equal portions, and these were blocked and cut in serial sections, and many of them stained by the Weigert hematoxylin method.

Right Side.—The uppermost levels showed degeneration of the fibers from the cortex anteriorly and posteriorly to the occipito-parietal fissure. In this region there was some necrosis of the cortex and subcortical substance, the section showing slight loss of tissue, particularly just subcortical.

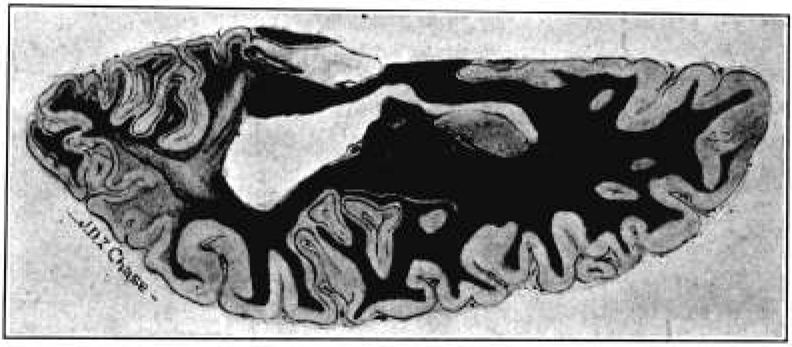

FIG. 1. Showing degeneration of white matter in occipital lobe and of the posterior part of the corpus callosum (right side).

In sections from a slightly lower level the degeneration was more apparent, and extended further posteriorly, although not entirely to the extreme posterior pole. There was no degeneration of the white matter on the median aspect. With a two thirds objective there were seen much perivascular distension, and some round-cell infiltration, in the degenerated area, and these changes were less where the degeneration of the fibers was less intense.

At a still lower level the degeneration extended forward into the ascending parietal convolution, and also to the posterior limit of the convexity.

Still lower, the ascending parietal convolution was slightly involved in its posterior aspect. The degeneration began just posterior to the interparietal fissure, and extended to the occipital pole. The median portion of the occipital lobe was not involved.

In sections from block 4 the white matter just posterior to the ventricle was degenerated, as well as the external surface of the occipital region, extending to the occipital pole, although here it was less intense. The fibers coming from the calcarine region at this level stained well, as did also the inferior longitudinal fasciculus, although it was smaller than in the normal brain. At this level the posterior part of the corpus callosum stained less distinctly than normally. The posterior horn of the lateral ventricle at this level was markedly dilated.

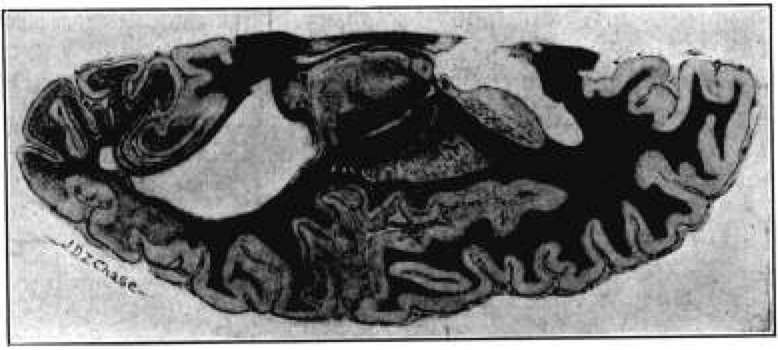

FIG. 2. Showing degeneration of the white matter of the occipital and second temporal convolutions of the optic radiations and the inferior longitudinal bundle (right side).

In sections still lower from this block the same condition existed, except that the degeneration extended to the inferior parietal lobe. The inferior longitudinal fasciculus, which was much smaller than normal, was degenerated at this level. The optic radiations and the tapetum were also degenerated. The median surface stained normally.

In sections from block 5 there was, in addition to the condition just described, involvement of the second temporal convolution to a slight extent, in its posterior portion. The splenium stained poorly, while the tapetum and inferior longitudinal fasciculus appeared to be smaller than normal. The forceps was smaller than normal, and the optic radiations were degenerated. The median surface was normal.

In sections from block 6 there was a small area of degeneration in front of the anterior occipital fissure of Wernicke. The optic radiations of Gratiolet, the tapetum, and the splenium were all degenerated.

In the lowest sections that were cut and stained the degeneration of the white matter did not extend in front of the interoccipital fissure. The inferior longitudinal fasciculus was normal. The internal capsule and basal ganglia showed no abnormality, and the pons, at the level of the red nucleus, which was included in the lower sections, stained normally. The foot of the peduncle showed no degeneration. The zone of Wernicke was not degenerated and the pulvinar and geniculate bodies appeared to be normal.

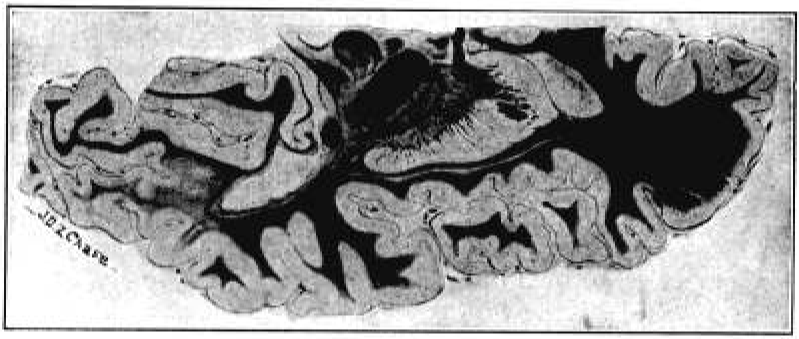

FIG. 3. Showing degeneration of the white matter in the occipital lobe posterior to the interoccipital fissure (right side).

Left Side.—Sections from the uppermost levels (blocks 1

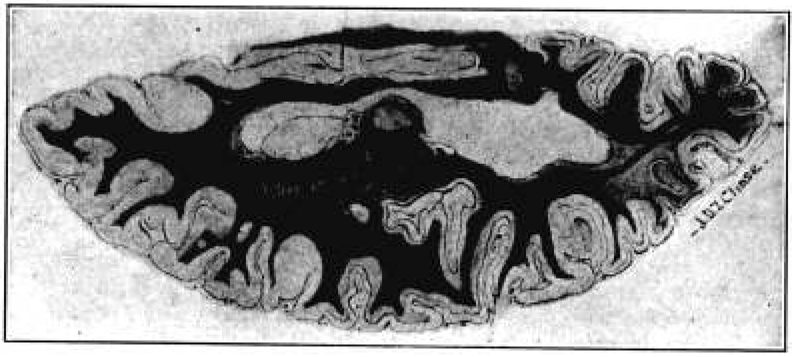

FIG. 4. Showing degeneration in the posterior ventricular region, external portion of occipital lobe, inferior longitudinal fasciculus, corpus callosum, and optic radiations (left side).

and 2) showed only a very slight degeneration in the occipital region. There was also some slight perivascular distension, and infiltration of round cells.

In sections from block 3 the degeneration was very slight, and situated in the external portion of the occipital region. Degenerated fibers could be traced from this region towards the corpus callosum. The inferior longitudinal fasciculus stained normally.

Sections from block 4 were degenerated in the posterior ventricular region. The degeneration in the occipital lobe at the convexity, while apparent, was slight. The median surface was normal. The degeneration did not extend into the parietal region. At this level the optic radiations, and the inferior longitudinal fasciculus, were slightly degenerated, and the posterior portion of the corpus callosum markedly so.

In sections from block 5 the degeneration was still apparent in the external portion of the occipital lobe. The tapetum and inferior longitudinal fasciculus were smaller than normal, but stained well, and the posterior horn of the ventricle was dilated.

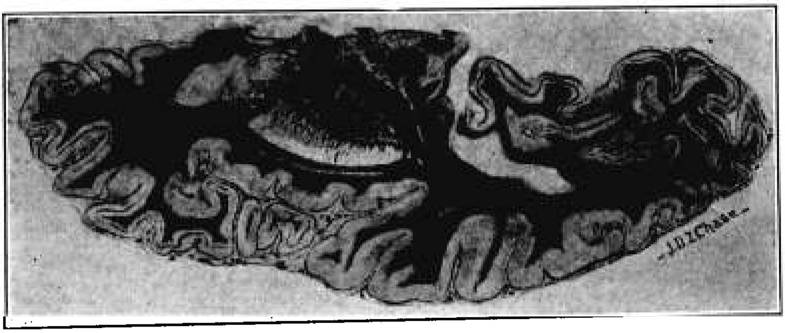

FIG. 5. Showing degeneration limited to the external portion of the occipital lobe (left side).

A little lower the sections showed degeneration limited to the external occipital region. The external capsule, the inferior longitudinal fasciculus, and the tapetum were normal at this level, excepting that the latter two were smaller than in the normal brain. The lower temporal lobe was slightly implicated in its posterior portion.

In sections from block 6 the degeneration was still apparent in the external occipital region, but otherwise the sections appeared to be normal, including the basal ganglia, the foot of the peduncle, the pons, and the zone of Wernicke.

The optic chiasm stained normally (Weigert method), as did also the right and left optic nerves.

Microscopically the cells were unchanged in both parietal regions (thionin stain). There was a moderate degree of perivascular distension. The blood vessels were thickened within the brain substance, and a few compound granular cells were found around the blood vessels. The pia was much thickened on both sides, but less so on the left. On the right side hemosiderin was found in the pia.

In the paracentral regions the pia was thickened slightly, and a mild degree of perivascular distension was observed. The cells stained normally.

In the calcarine region there was slight perivascular distension, but the cells stained well. The pia was slightly thickened, the more so on the right.

Sections from the necrotic area in the right occipito-parietal regions showed the presence of a few compound granular cells, a number of spider cells, marked perivascular distension, thickened blood vessels, and some perivascular infiltration. A few of the perivascular spaces contained blood pigment, probably hemosiderin.

Sections from the pons and medulla oblongata and the spinal cord, stained by the Weigert method, showed no degeneration.

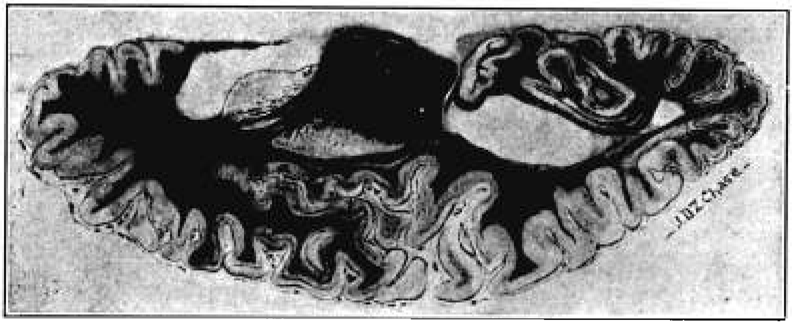

FIG. 6. Showing slight degeneration of external part of the occipital lobe (left side).

Cells in the anterior horns of the cervical and lumbar regions were slightly diseased. The Nissl bodies were atrophied, and the yellow pigment was increased. Some had lost their prolongations, but there were many normal cells.

SUMMARY: A man of 55, at the time of his admission to the home, was blind; was totally unable to designate the position of the limbs; could not locate touch anywhere; could not recognize objects by the sense of touch; and his touch and temperature senses were imperfect in the left hand. The left hand, although capable of some reflex acts, could not be moved voluntarily. The right hand was apraxic, and apraxic phenomena were present in chewing and walking.

The autopsy revealed the presence of degeneration of the white matter of the right occipital and parietal regions on the convexity, and the posterior portion of the temporal lobe, the calcarine region remaining intact. The inferior longitudinal fasciculus and the optic radiations were degenerated on the right, and probably though less markedly on the left. On the left side there was degeneration in the occipital and temporal regions to a much less degree, leaving the median surface intact. The corpus callosum, in its posterior portion, was degenerated. Elsewhere the brain was apparently normal.

The process was probably the result of arteriosclerosis which caused degeneration in the white matter from deficient nutrition. The necrosis of the right convexity was probably of the same origin.

The difficulties of explaining the symptoms presented in this case are very great, on account of the absolute loss of the sense of position and movement, the loss of the ability to locate tactile impressions, and the total blindness.

Was his utter lack of knowledge of the position of his body—for he could not tell whether or not his limbs were moved, or whether he was standing or sitting—due to a loss of muscle sense, or was it related in some way to the blindness?

Pathologically the general loss of muscle sense was not explained. The lesion in the right parietal region would account for the sensory change on the left side, but absence of involvement of the left parietal region leaves this symptom on the right side unexplained, for, according to von Monakow, the parietal region is the center for the muscle sense and the sense of location.

The absence of lesions sufficient to explain in the usual way the general loss of the sense of position of the entire body leads us to suspect that the destruction of the fibers going to the visual centers may play some part in causing it.

A possible analogy is seen in those cases of mind or psychic paralysis (Seelenlähmung) in which, with the eyes open, a limb is moved freely, but when the eyes are closed the limb acts as if dead; that is to say, when the optic element is cut off, or poor, the movement is disturbed, and the location of the limb is not recognized. An example of this was present in Liepmann's patient who, with the eyes closed, lost all knowledge of the position of the right arm. This is not unlike the facts presented by my patient, whose entire body, on account of his blindness, was constantly in the same condition as was the right arm of Liepmann's patient, when he closed his eyes.

Or, was the loss of the sense of position of the body a form of disorientation, analogous to that which is observed in mind- or psychic blindness (Seelenblindheit)? Against the acceptation of this latter view, however (in spite of the fact that the post-mortem lesion was that which is commonly found in mind-blindness), is the absence of the characteristic symptoms of mind-blindness. The mind-blind patient sees objects, and has light impressions, but is unable to recognize the nature of the objects which he sees.

In my case, as the examination of Dr. Posey and myself clearly demonstrated, the man was totally blind. The report of Drs. Webster and Terriberry does not convince me that he was suffering, at that time, from mind-blindness. All the symptoms which he is reported to have presented could be readily accounted for by pure blindness with slight preservation of light perception.

It is possible that there might have been some light perception preserved after his admission to the home, in view of the fact that when the hand was suddenly placed before his eyes, while he was walking, at times he hesitated or stopped, according to the observation made by the orderly; but this observation was never confirmed by myself, although frequently tested for, and he was often seen to run into things and had injured himself in that way. In mind-blindness there is a loss of the recognition of the nature of the objects, with preserved sensation, and this is not true in my case.

Apraxia.—The movements which I believe to be probably due to apraxia are as follows.

1. The curious disturbance of the gait. The patient took short, rhythmical steps, usually to one side. That this distorted gait was not due to his blindness is probable, as he did not seem to have any fear of bumping into things, and the peculiarity of the gait continued in spite of his being assured that every precaution was taken to prevent his bumping into objects.

2. The false movements. In eating, for example, in an effort to place to his mouth a banana, he would invariably place it first upon his chin. This was not due to ataxia, as there was not the slightest appearance of this symptom in any of his movements.

3. Or, if a pen were placed in his hand, and paper before

him, and he was asked to write, only hacking movements from right to left resulted (apraxic agraphia?).

4. A watch was placed in his right hand, and he was requested to put it to his ear. He recognized that it was a watch, though not by touch, but instead of putting it to his ear (although he sometimes succeeded) he usually placed it to his mouth, believing that he was holding it to his ear, at the same time going through the movements of chewing.

5. When asked to touch his ear with the right hand, he made groping movements in the air, or grasped his knee, or his coat, and pressed tightly, believing that he was touching his ear. Or, he would stand up and make one or two steps, still believing that he was touching his ear.

6. Apraxic movements of the muscles of mastication.

Some reserve is necessary in deciding whether or not these movements were apraxic; as the studies could not be carried beyond a certain point, or in great detail, on account of the loss of the sense of position, the blindness, the loss of perception of the nature of objects by touch, and the perseveration present in the left hand. These symptoms, therefore, do not entirely accord, in all respects with the definition of apraxia given by Liepmann, for these reasons.

According to Liepmann's definition of apraxia, an object is recognized, its use known, the limbs themselves are readily moved, so that the innervation of each limb is intact, but purposeful movements (Zweckbewegungen) of the affected limb are lost.

The amorphous movements of G. in walking and attempting to touch the nose, however, correspond to similar movements in Liepmann's case.

The false movements in eating, in the case of G., which were present in Kleist's (16) case, and possibly the amorphous movements of the hand, in the effort to write (if this was not pure agraphia) may be classified under the head of "False Movements."

Agnosia.—Can the element of agnosia be eliminated in explaining the nature of these movements?

When my patient was eating a banana he recognized the object, although not by touch, but by a psychical process. He understood that, in order to eat it, he must put it to his mouth; but when he made the effort to place the hand to his mouth he never succeeded, the banana invariably landed on the chin.

In attempting to place a watch to his ear he recognized that it was a watch, not from hearing the tick, or from the sense of touch, but from the fact that it was a watch that the examiner would naturally place to his ear for him to listen to. In other words, he recognized the nature of the objects which he attempted to use. Moreover, he could use his right hand for reflex acts. He therefore knew the nature of the object, its use, and was able to move the right arm, but he could not use the object properly. It seems to me, therefore, that the false movements that occurred under these circumstances were apraxic.

The explanation of the symptoms in my case is scarcely to be found on the basis of agnosia; at least my conception of agnosia. As I understand it, agnosia is a loss of the understanding or the perception of the nature of things, while sensation is preserved, an illustration of which may be seen in the loss of sensory memory forms (Wernicke), or a loss of the connections between sensations and memories in mind-blindness, mind-deafness, and mind-paralysis, which, according to Hartman (12), are expressions of agnosia.

Nodet (36) defines agnosia as a disturbance of secondary identification, with persistence of the primary identification, and Liepmann, Hartman and others also looked upon agnosia as a failure of recognition (Erkennen) with preserved sensation. The identification of the fresh impressions with the memory forms does not occur, either on account of the loss of the latter, or on account of a hindered connection between both of them (Liepmann).

In other words, agnosia is a difficulty of recognizing cortical sensory impressions, and a difficulty of connecting these impressions with a mental picture. For example: One takes an object in the hand in which all forms of sensation are preserved; recognizes its shape, physical, and other properties, and can describe them fully; but is unable to recognize the use of the object, or its name. This failure of recognition is due to an interruption between the memories for objects, and the sensory centers in the cortex.

These are not the conditions present in my case. There were, on the left side, distinct sensory changes manifested in the disturbance of the temperature sense, the sense of location, the sense of movement, and the muscular sense; while on the right side the sense of location, the sense of movement, and the muscular sense were altered.

Felix Rose (43), who believed that my case was one of agnosia, stated that in agnosia the confusion of movements is based upon the fact that the patient takes one object for another; for example, a toothbrush is used as a pencil. He admits that in motor and ideatory apraxia the substituted movements are observed, but the association between the logical act, and the executed act is evident; for example, the patient brushes the beard with the toothbrush. Rose believed that agnosia produces especially the substituted movements, while this is the exception in motor apraxia.

In the case of G. there is no analogy with these statements of Rose.

Substituted movements occurred when my patient made an effort to touch the ear, for example, when the right arm was used in a groping manner, as illustrated in Liepmann's case. While he placed the watch to his mouth instead of to his ear, it was not because he did not recognize what the watch was. Moreover, in the case of G. the primary identification was at fault, a condition the opposite to that described by Nodet and others, who state that in agnosia primary identification is preserved.

Apraxic Movements.—While the apraxic movements could not be studied in detail, for the reasons above enumerated, those which I claim for my patient are similar to those described in at least some of the cases in the literature.

In Liepmann's (21) case the apraxic movements consisted, among others, of amorphous movements which were represented as follows: If Liepmann's apraxic was asked to point to his nose, the request was followed by a strained position of the arm on the right side, nodding of the head, or repeated bowing, the patient thinking all the time that he was touching his nose. This was analogous to the movements observed in my case, in which the patient, in an effort to touch his ear, groped in the air aimlessly with his right hand, or grasped his knee, or stood up and made one or two steps.

When Liepmann's patient was asked to point to the left hand with the right, he nodded " yes," and lifted an ink well in front of him. He could not make a fist with his right hand, distorted movements following instead. He could not dress or undress himself (which was true in my case), and many special acts, as well as imitation movements, were unsuccessful.

The similarity of the movements in my case to those of Hartman's patients is striking. In his cases the direction of the movements was missed. When the patient attempted to touch the right extremity with the left he failed, inappropriate, distorted movements occurring as to direction and form. Taking nourishment by the mouth was performed very slowly, improperly, or done in stages.

In Pick's (40) cases the disturbances of movement were somewhat different, and may be looked upon as expresions of ideomotor apraxia. For example, a patient brings a lighted match near a candle correctly, lights it, and then finally blows it out.

Again, the patient handles a pistol as if it were a musket; or, if given a cigar and a matchbox, the patient evidently recognizes the cigar, but opens the matchbox, sticks the cigar into the open end, and presses upon it as if it were a cigar-cutter. He then rubs the cigar on the side of the matchbox as if it were a match; but finally the movement is made correctly.

Or, a watch is shown to the patient, and he is asked what it is. After many questions, he takes it in his hand and raises it to his lips. This resembles the movements in my case.

Bonhoffer's (4) patient struck the cigar on the matchbox.

D'Hollander's (14) patient, when requested to strike a match, grasped the matchbox in his left hand, took out a match with the right, hesitated, placed the match in his mouth, then finally replaced it in the matchbox again. His case was one of paralytic dementia in which the autopsy revealed the characteristic lesion of this disease without focal lesions.

In Kleist's (16) case of cortical, or innervatory, apraxia, the patient was requested to sharpen a pencil with a penknife. He took the pencil in the left hand, grasped the knife with the right, sometimes with the back of the blade, and sometimes with the cutting edge uppermost, and supinated the forearm so extremely that the knife was held with the edge, instead of out and under, in and down. When he had succeeded in pronating the arm sufficiently in the effort to sharpen the pencil, the necessary extension and flexion of the arms brought the position back to supination. He then pressed and scraped the pencil, then shook his head with an expression of distress. At another time the patient made an effort to cut a piece of paper with a pair of scissors. Instead of placing the thumb and forefinger in the openings of the scissors, he held the scissors between the thumb and forefinger, and pressed these against each other. Later the thumb and finger were placed in the openings, but in the effort to open them they slipped out, and he held the scissors as before. Instead of opening them he pressed the handles together. Once or twice he succeeded in making the cutting movements, but then lapsed again into the movements just described (innervatory apraxia).

In Kleist's cortical, or innervatory apraxia, the disturbance is manifested in all forms of movement. There is an incapacity to perform those movements which arise from special memories; in other words, memories acquired by practice. Antagonistic, or useless movements appear. Partial acts are undisturbed, but the preparation of the act is implicated in its innervation. The motor memories are involved (auto-kinetism), not alone the sensomotorium—differing from Liepmann's apraxia in which the sensomotor " Eigenleistungen " is intact—and therefore not due to any disturbance of the movement formula.

In Strohmayer's (46) case the patient put his hand, instead of the spoon, into the soup, or cut with his fork or the back of his knife, while recognizing the objects and their use.

Bonhoffer's patient, in making an effort to shut the door, moved the hand in an apraxic manner. He held the key in his hand, making thrusting movements with it instead of the proper ones. He was unable to write certain letters, although he knew them, but produced figures without character. The latter Bonhoffer believed was an apraxic agraphia.

In Lewandowski's (17) case of progressive paralysis, the patient was able to make only three movements with the left arm, *i. e.*, to the back of the ear, to the mouth, and rubbing movements. These, however, were probably not true apraxic movements.

In a second case reported by Lewandowski (18) there was apraxia of the eyelids. The patient was unable to close the eyes on command, neither one at a time, nor both together. This was, according to this observer, independent of a slight left facial paralysis.

It will be seen that there is a great variety of abnormal manipulations (" Handlungs," Liepmann; " Agierns," Abraham (2)) of objects, which are described under the head of apraxia. These may be divided into (1) manifestations of ideomotor apraxia (ideokinetic, of Liepmann, formerly motor apraxia); (2) ideatory apraxia (ideomotor apraxia—Pick); (3) innervatory apraxia (Kleist).

Von Monakow (34) classifies apraxia as follows: (1) Bilateral apraxia associated with right-sided hemiplegia; (2) agnosia, or sensory apraxia; (3) amnesic apraxia, as in cases of progressive paralysis; (4) unilateral apraxia, as in Liepmann's case; (5) ideatory apraxia (Pick). He states that in apraxia movements remain undisturbed, or are only slightly involved, in (1) breathing, swallowing and eating; (2) in elementary movements of orientation, such as turning the eyes and head in the direction of an irritant, when there is not a concomitant central optic, or other trouble; (3) in gross reflex acts, as in movements of defense; (4) locomotor movements, as sitting up, or walking; (5) in simple acts (moderately free from involvement); and in (6) simple automatic movements, such as unbuttoning a button.

According to Liepmann, motor apraxia affects single limbs, and is rarely bilateral (cases of Hertzog (10), Liepmann, Strohmayer, and possibly my own case), in contrast to ideatory apraxia in which both sides may be affected. Motor apraxia appears in simple acts, such as putting out the tongue, or making a fist. This is not true of ideatory apraxia, which manifests itself in complicated acts especially. In motor apraxia the limbs do not obey the psychical wish. In ideatory apraxia there is a failure of the psychical conditions for the correct completion of the act, but the limbs respond properly.

In motor apraxia the motor memories for the extremities are preserved, but they are insufficiently connected with the other cortical fields. The ideatory process and the motor memories are separated. In ideatory apraxia the motor memories are intact, but the ideatory scheme is at fault.

Resorting to explanation by formulas, Liepmann (19) employs Wernicke's scheme.

According to Wernicke, the sensory centers (*S*) perceive sensory impressions. *SA* represents the psychosensorial pathway, so that at *A* there develop end memories (Ausgangvorstellung), which are discharged at *Z*. *AZ* is the intrapsychic pathway. *Z* represents the end memories (Zielvorstellung). These excite the motorium through *ZM*. *ZM* is the psychomotor pathway.

Liepmann modifies this formula so that *Z* represents the chief end memories (Hauptzielvorstellung; Erfolgsvorstellung). From

Z there go a number of paths to z^1, z^2, z^3, z^4, and from each of these there goes a path to M, the motorium. He then substitutes for the M, J the innervation. $M =$ the motorium which becomes excited to new activity through the action of J, the innervation.

The path Z to z must be reckoned as the intrapsychic region, so that the horizontal line G (see diagram A) would represent a division of the intrapsychic and psychomotor regions, not the vertical line G in diagram B.

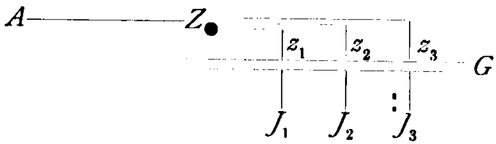

Diagram A.

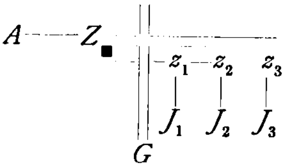

Diagram. B.

Each memory for a partial act (z^1, z^2, z^3, z^4, z^5) is called a "Zwischenzielvorstellung." From each z there goes out an innervation J which results in a movement or action. Failure of reaction in inattention, and the early disturbance of purposeful movements (Handlung) in brain diseases, show that the Z's give off false z's, but the proper connection between the z's and the J's is maintained. The J's correspond constantly to the z's, and this is the condition that Liepmann believes is present in the Apraxia of Pick.

Summarizing the whole matter, Liepmann states that purposeful movements (Handlung) in a normal individual, are the result

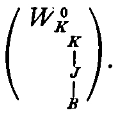

Diagram C. Memory Complex.

of a memory complex made up of partial memories (see diagram C); W, the direction memory (Richtungsvorstellung); o, optic memories; K, general kinesthetic memories; k, limb kinesthetic memories; J, the innervation; B, the external movement. Purposeful movements depend upon a number of these complexes which replace the z's in the formula.

The same disturbance can be referred to the lapse of these complexes as to the lapse of the memories themselves. The subjective result would be that instead of a proper $W^0_{K_k}$ a false $W^0_{K_k}$ would appear, or in a false place. The objective results would be that a false J and B would appear, but always corresponding to the $W^0_{K_k}$. The innervation fails proportionately to the ideation, resulting in ideatory apraxia.

But, if there is a separation within each complex, so that everywhere the J's, or that J with the immediately adjoining k, do not any more correspond with the rest of the memory complex, then there results a disturbance of movement which is motor apraxia.[1]

In other words, Liepman believes that, in motor apraxia, there is a dissociation of the kinetic memories by reason of an organic focal lesion. The psychic element and its associations are preserved.

Kleist (15) modifies Liepmann's formula so that each direction memory contains a general kinesthetic element (K), as well as optic element (O), claiming that in Liepmann's formula the K is psychologically incorrect, being properly only a component of each direction memory, and should be written W^0_K. He believed that the partial memories are not a part of the chief end-memories, but a part of the complex of the total movement memories. His formula is as given on next page:

Lesions of Apraxia.—In Liepmann's (23) case, which is the foundation for the newer conception of apraxia, numerous connections between the left central convolutions and the cortex of the frontal region were severed by a subcortical focus in the frontal brain. A subcortical focus in the left parietal region implicated the connections from the occipital and temporal regions with the central convolutions, and destruction of the corpus callosum, as far as the splenium, separated the central convolutions from the entire right hemisphere. In the right hemisphere there was also a focus where the gyrus supramarginalis joined

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[1] This is an incomplete résumé of this phase of the subject, and reference should be made to Liepmann's "Ueber Storungen des Handelns, etc.," by any one wishing to study the subject more fully. Some of the matter relating to the elucidation of the subject by formula is translated freely from the original.

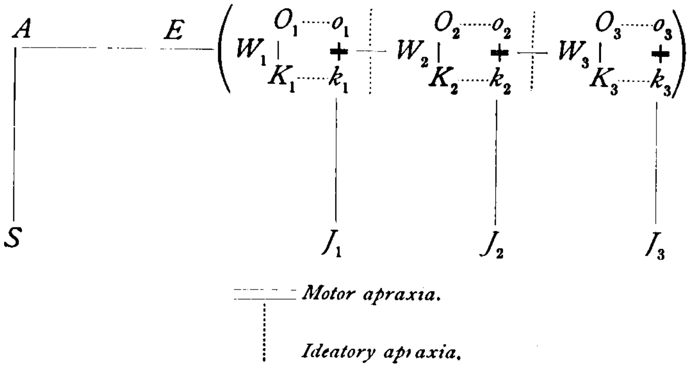

Diagram D. Kleist's modification of Liepmann's formula.

the gyrus supra-angularis in the right hemisphere; and a second focus that destroyed most of the fibers from the left face, arm and leg centers.

Hartman (12) reported three cases of apraxia, with post-mortem examination. In the first there was a tumor in the left frontal region, which spared Broca's region and the neighboring white matter, and extended to the anterior thalamic levels in the left corpus callosum. In the second case there was a tumor which destroyed the corpus callosum from the level of the anterior commissure to the posterior end, and which did not extend to any part of the brain. In the third case there was a hemorrhage in the second right frontal convolution the size of a walnut.

In Van Vleuten's (52) case of left-sided motor apraxia there was a cylindrical tumor involving the entire corpus callosum, the left gyrus fornicatus, and part of the frontal brain. The cortical white matter was not involved.

More recently a case was reported by Liepmann (27) and Mass, with left-sided agraphia and apraxia, in which there was a cyst that began in the left upper frontal region and in the gyrus fornicatus, and extended posteriorly in the latter to the paracentral region. The left half of the corpus callosum was entirely implicated in the left hemisphere. There was also a focus the size of a pea that interrupted the pyramidal tracts. In the right cerebellar peduncle there was a focus the size of a lentil, and one in the pons outside of the lemniscus, and one in the right thalamus.

In Von Bechterew's (5) case there was a circumscribed lesion in the left hemisphere in the middle part of the posterior central region, and in the posterior part of the gyrus supramarginalis.

Strohmayer (46) found in his case of apraxia a lesion in the lower parietal region, where there was an extensive loss of substance which reached forward to the posterior central convolution. It extended below to the fissure of Sylvius, and above as high as the anterior parietal fissure. The vertical section at the posterior end of the splenium showed that the focus spared the white matter of the optic region, and that parts of the forceps major and the superior longitudinal fasciculus were involved. The striking similarity of Liepmann's case is commented upon.

In Pick's (42) case of senile dementia with apraxia there was atrophy of the brain accentuated in both frontal lobes, and the left inferior parietal lobe. It was less severe in the right inferior parietal lobe, and both temporo-occipital lobes. Nothing abnormal was observed in the central convolutions, the superior parietal lobe, the cuneus and precuneus. This case is believed to have been the result of disturbance of the association centers of Flechsig.

Westphal (54) recently reported a case of motor apraxia in which the autopsy showed the presence of internal hydrocephalus, and several foci of softening in the right frontal and occipital regions, and in the left optic thalamus.

Abraham (2) described a case of total aphasia, mind-deafness, apraxia, agraphia and alexia, with autopsy. The brain was atrophic, especially in the post-central regions. The occipital lobes were relatively preserved. The lateral ventricles were distended.

The usual lesion of paralytic dementia was present in D'Hollander's (14) case of aphasic apraxia, but no focal lesion was found. There was some hydrocephalus.

Von Monakow (34) has recently reported two cases of apraxia with autopsy. In one apraxia was a partial symptom in a case of progressive sensory and motor aphasia. A tumor was found in the corpus striatum compressing the entire region supplied by art. foss. Sylvii. The cortical speech centers, central convolutions, and the corpus callosum were intact. He looked upon the case as an example of apraxia due to a subcortical lesion in the central ganglia.

In a second case the apraxia was a transient symptom, occurring in a case of sensory aphasia, and the autopsy revealed the presence of a lesion of the right central ganglia, and an infarct of the posterior third of the temporal lobe, implicating the gyrus supramarginalis.

A study of these facts shows that a variety of lesions have been described in cases exhibiting apraxic symptoms, and the lack of uniformity in the lesions described is striking.

What seems fairly well demonstrated in all this data is that the destruction of the corpus callosum plays a distinct part in the causation of left-sided apraxia, probably by cutting off from the right frontal brain the influence of the left frontal brain. The predominance of the left frontal brain over the right side has been demonstrated by Liepmann in cases of left-sided apraxia in right-sided hemiplegias, and dyspraxia of the left hand in right-sided apraxia and aphasia.

Manfred Fraenkel (8) described a case of mirror-writing and apraxia of the left hand with right hemiplegia, and came to the conclusion that the brain centers on the right side were, to a certain extent, under the control of the left side of the brain; that the left cerebral hemisphere directed not only speech, but "Handeln"; that the right cerebral hemisphere did not contain an independent center for movement memories; and, finally, that it was therefore closely related to the lower centers.

Rothman (45) has made similar observations in right-sided hemiplegia. He believed that the precedence of the left side of the brain for manipulations was demonstrated.

Liepmann and Mass, in analyzing their recent case, agreed pathologically with Hartman's second case, and Van Vleuten's case, *i. e.*, that the left hemisphere exercises a directing influence upon purposeful movements of the left hand; and that destruction of the corpus callosum causes a localizing symptom, *i. e.*, dyspraxia of the left hand. Liepmann and Mass claim that it amounts to the same thing whether the interruption of the corpus callosum separates the left hand from the memory centers of the left hemisphere, or, if the corpus callosum is a direct pathway for impulses to the sensomotorium of the right brain. Whether the chief rôle is the separation of the left hand center (Liepmann) or of the left frontal brain (Hartman), Liepmann believes the differences in opinion disappear if, as is probable, the kinetic memories of the sensomotorium of the upper extremity are in the middle frontal convolution.

Van Vleuten's case showed an intense apraxia of the left, and relative eupraxia of the right, hand. In this case the corpus callosum was destroyed, excluding part of the splenium.

In Hartman's case there was grave apraxia in the left hand, and a suggestion of apraxia in the right hand. The corpus callosum in this case was also destroyed, including the splenium.

In Liepmann and Mass's case the corpus callosum was destroyed, excluding the splenium. These observers believed that this demonstrated that the preservation of the posterior fourth and fifth of the corpus callosum does not guarantee eupraxia of the left hand.

Liepmann and Mass do not doubt that the splenium plays a rôle in the conduction of impulses from the left to the right hemisphere. Van Vleuten's case showed this, and Liepmann and Mass's case also showed that the posterior portion of the corpus callosum is of less significance for eupraxia in the left hand than the middle portion.

Hartman believes, with Liepmann, that there is a center in the frontal lobes for the mechanism of motor cerebral activity, analogous to Broca's region in aphasic troubles, and that destruction of the left frontal lobe causes apraxia. The right frontal lobe needs the coöperation of the left brain for the outflow of purposeful movements. Defect in the right frontal region (right mid-frontal) causes partial conduction-apraxia of the left side, with preserved movement memories (Hartman's third case).

Felix Rose (44) has recently reported a case of transient bilateral apraxia of the muscles of the inferior distribution of the facial nerve, of movements of the tongue and the jaw, and of the left hand, in a patient suffering from Jacksonian crises of the face and right arm. He believed that there was a diffuse meningo-encephalitis (no autopsy) which caused these symptoms, and concluded that it must be deduced that while the right hemisphere exercises no influence on the limbs of the right side for unilateral movement, and certain bilateral manipulations, for certain bilateral habitual motor functions, particularly of the muscles of the jaw, tongue and face, the integrity of the two hemispheres and their association pathways is necessary.

In discussing the significance of the corpus callosum in relation to apraxia, von Monakow (34) stated that for the preservation of praxic movements the integrity of the corpus callosum was not necessary, and in support of this opinion cited a case of intense hydrocephalus, in which the corpus callosum was only rudimentary and the majority of the fibers of which were degenerated. The patient could not only use both hands alternatingly in housework, but could also write. There was no apraxia, in spite of the fact that the white matter of the left parietal lobe and of the left temporal lobe, was defective to a great extent.

Van Vleuten claims that his case supported the view held by Marie, that apraxia is due not only to a destruction of a movement idea, but to a separation of the corpus callosum connections, between the material basis of the movement idea in the left hemisphere and in the right hemisphere. The special teaching of his case was that dyspraxia could be caused by a lesion destroying simply corpus callosal fibers. In van Vleuten's case the right hand was not apraxic, and he believed, therefore, that the left-sided apraxic center has not any influence over the sensomotorium of the right side.

Von Bechterew (5) believed that apraxia was localized in the parietal lobe, and was due to a loss of movement memories.

Pick's view in his case, with autopsy, was that there was a disturbance of the association centers of Flechsig.

Liepmann's opinion is that apraxia results from a dissociation of the motor centers from many regions of the brain. And further, that the entire memory of complicated acts is not confined to certain places in the cortex, either in the central or parietal convolutions. Optic, tactile, kinesthetic, as well as acoustic, elements, he states, figure in the general memory, and a circumscribed focus could scarcely cause the loss of the entire movement memory. The movement memories are collected, he believes, in the left sensomotorium, and there associated with the other territories of both hemispheres.

Margulies (28) held, on the contrary, that apraxia could be localized, for, he states, if inability to accomplish simple acts is present, localization at once becomes evident. In disturbance of complicated acts the physiologic point of view indicates a primary localization. Bonhoffer believed the apraxia in his case was an expression of a focal lesion.

Kleist, in his recent case, claimed that the trouble was in the innervation, and was a fault of the so-called sensomotor " Eigenleistungen," thus differing from Liepmann's case in which the latter was intact.

Von Monakow (33), in an early opinion, did not subscribe to the view that apraxia could be a permanent symptom of lesion of the parietal lobe, and he believed that it was related to cortical ataxia, as motor aphasia is related to disturbance of articulation. It is due, he thought, to a series of variable and general factors, such as is seen in aphasia, and is the result of " diachisis."

In a more recent opinion von Monakow (34) stated that, in the pathology of apraxia, the region supplied by the left art. foss. Sylvii is the locality of predilection. He believed that the left gyrus supramarginalis plays an important part in the genesis of apraxia, and that in unilateral apraxia of long duration, though not necessarily persistent, there is local or diffuse involvement of the P_2, T_1, and probably also of the corpus callosum.

Intellectual Element in Apraxia.—Marie (29) believes that apraxia is due to an intellectual deficit, and that it really is an aphasia of feeble intensity. He claims that the patient does not understand the orders given, nor has he the exact notion of the gesture corresponding to the order given.

While Liepmann (26) acknowledged that focal disease causes weakening of the intelligence, he looked upon this as a result of the lesion causing apraxia, and not the cause of the apraxia itself.

Van der Vloet (50) does not subscribe to Marie's opinion, and, as a result of the study of 14 cases, believes that dementia and apraxia are independent of each other.

In this connection the recent views held by Abraham (2), apropos of a case reported by him, are interesting. He believed that the psychic element plays an important part in apraxic phenomena, and that this component has not received the attention it deserves in the study of this subject. The symptoms which, in his case, he believed proved this were the following. His patient, in putting on his coat, dropped it accidentally. He then attempted to put the right hand into the sleeve, but could not. Finally, he made a fold in the coat, put his hand through it, and was satisfied.

In another instance he put his trousers on wrong side about, and when his attention was called to it he turned around, believing that this corrected the mistake. The last act represents a psychic deficiency. Abraham questions whether all cases of motor apraxia have not more or less psychical disturbances.

The significance of the psychical element in apraxia has also been emphasized by Margulies (28), who believed that psychical symptoms are manifested in ideatory apraxia, if an incomplete, partial motor apraxia or agnosia is present, apropos of Pick's cases, and of cases of amnesic aphasia in localized senile atrophy.

My own view is that there is always present either a disturbance of intelligence, or agnosia; for example, the apraxic believes he is performing an act properly, when in reality it is being performed in an apraxic manner. He does not recognize the incorrect result, even when he sees it. There is here more than a suggestion of a loss of perception connected with false interpretation of optic and movement impressions. Whether this is psychic, or a manifestation of agnosia, is a question. It is possibly both, and, as Liepmann claims, is the result of the lesion causing apraxia, rather than the cause. The fact that apraxic symptoms have been observed in cases of senile and paralytic dementia (Pick, d'Hollander (14), Abraham (3), Lewandowski (17), Soutzo and Marbe (47), and Marcuse (31)), and in post-epileptic conditions (Pick (40) and Oppenheim (38)) is suggestive in this relation.

Psychic, or Mind Paralysis (*Seelenlähmung*).—The behavior of the left hand in my case resembles, in some respects, the " Seelenlähmung" described by Nothnagel and Bruns. My patient could not move the left arm in voluntary acts demanded of him, and it was held in the same position all day long, with the exception of occasional movements of a voluntary reflex character. For example, he could perfectly well put his left hand in his hip-pocket, take out his handkerchief, wipe his nose, and return the handkerchief to his pocket, and did so on several occasions.

In Bruns's (6) case of sensory aphasia, alexia, agraphia, slight paraphasia, and right hemianopsia, with lowered touch, pain, temperature and position senses on the right side, the right arm was incapable of spontaneous involuntary movements, but could be used in pure reflex or unconscious voluntary movements. Moreover, by practice other movements were possible.

It made no difference if these movements were controlled by the eye, ear or hand. My case differs from this in that the arm was not helped by practice, or assistance from the other hand, or the hand of the examiner. Bruns believed that in his case the symptoms were due to softening of the left parietal and temporal regions, as well as the gyrus angularis, and the posterior part of the internal capsule. The rest of the hemisphere, especially the central convolutions, was intact. He stated that disturbance of the sensory centers, and the subcortical fibers whereby the destruction of the fibers from these to special motor centers is occasioned causes an incapability to use the limb in voluntary movements. If, by reason of the breaking of these fibers, the psychical center has no longer an influence over movements, there exists a mind-paralysis for voluntary movements, although these may be unhindered reflexly. Therefore, it is the result of an unilateral destruction of the sensory centers and their association fibers.

Somewhat similar cases have been observed by Anton (1) and Bleuler (7).

Bruns concludes that this condition is a disturbance that arises alone from the falling out of the memories for movement.

Nothnagel, who was the first to use the term psychic paralysis (Seelenlähmung), believed it was due to a separation of the cortical fields for motor memories in the parietal lobe from the motor centers.

Liepmann subscribed to the view held by Monk and Nothnagel. Monk defined " Seelenlähmung " as the complete loss of sensory memories of a portion of the body, which, as Liepmann states, corresponds to the more recent view of the loss of kinesthetic memories, and to Meynert's motor asymboly, Nothnagel's " Seelenlähmung," Heilbronner's cortical apraxia, and Liepmann's loss of limb kinetic memories (Gliedkinetischenvorstellung), and therefore not a manifestation of apraxia. In Liepmann's case of " Seelenlähmung " the limbs could only be used skillfully in speaking and gestures.

Perseveration.—When, after some effort, my patient succeeded in grasping the hand of the examiner, he continued to grasp it with increasing pressure, which would persist as long as the hand of the examiner remained within his grasp. This symptom, without doubt, was an example of tonic perseveration.

Perseveration has been classified by Liepmann (19) as tonic, when contractions of the muscles persist; clonic, when there is an alternation of contraction and relaxation, without apparent cause; and intentional, when an act becomes repeated instead of the intended new one. Cases of this character have been reported by Veschidi (49), Vurpas, Kleist and Liepmann, and have been met with in verbal deafness, mind-blindness and cases of agnosia. According to Liepmann the rare cases of tonic perseveration are due to disturbance of the motorium itself. This opinion seems to be somewhat confirmed by the pathological findings in my case, in which the right ascending parietal convolution was in part degenerated; that is, providing we believe that the motor centers are not entirely confined to the precentral region.

Tactile Paralysis (*Tastlähmung*).—The failure to recognize the nature of objects by touch is difficult to explain in my case. The impairment of sensation on the left side, outside of the loss of the sense of position, and movement sense, sufficiently explains its presence on this side, but on the right side all forms of sensation were intact, except the sense of position, movement and localization.

There was no ataxia, in spite of the presence of sensory symptoms which usually cause this phenomenon. In the absence of lesions causing tactile paralysis, it is difficult to explain its presence unless we attribute some significance to the optic memories, in other words, it may be the result of a separation of the sensomotorium from the optic centers.

In Nicolauer's (35) case of apraxia and tactile paralysis (Tastlähmung) in the same limb, the question arose whether both symptoms were not due to a focal trouble in the right hemisphere, which involved the central convolutions, causing disturbance of touch and other sensations on the one hand, and which interfered with motor acts by injury to the conduction of the nerve impulses required for their proper performance, on the other hand.

The pathologic cause of the apraxia in my case must remain problematical, unless apraxia may be caused by lesions of the occipital and temporal lobes in which, perhaps, are lodged, in part at least, the memories for purposeful movements.

It occurs to me that perhaps the memories for purposeful movement are not centered in any one locality, though there may be a region where all the sensory impressions are congregated, but this is only a meeting-place, so to speak, for these impressions which have their cortical localization in the primary centers.

If I am correct in my opinion that my patient was not mind-blind, but actually cortically blind, the lesions of the occipital lobe, confined to the convexity and leaving the medial areas intact (including the calcarine region), do not coincide with the present-day view of the central localization of vision.

If it is true that vision is centered in the calcarine region, which was not degenerated in my case, why did he not see?

I am not prepared to make the statement that vision depends not only on the function of the calcarine cortex, but needs also the coöperation of the cortex of the entire occipital lobe, but I am willing to admit that this is a possibility which seems to be suggested by the postmortem findings in my case, for the inferior longitudinal bundle and the optic radiation were not entirely, or completely, degenerated.

In conclusion, it is worthy of note that in a number of the cases of apraxia with autopsy a certain degree of hydrocephalus has been found. What significance this may have is conjectural, and I shall at present go no further than to call attention to the possibility that the association of hydrocephalus and apraxia may be more than coincidental.

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